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AN ESTIMATE OF ADULT MORTALITY IN THE UNITED STATES FROM PASSIVE SMOKING; A RESPONSE

Dear Editor:

Wells (1988) estimates that exposure to environmental tobacco smoke (ETS) causes 46 000 deaths per year in the U.S.; 3000 from lung cancer, 11 000 from other cancers, and 32 000 from heart disease. These estimates are scientifically unjustified. Far too much faith is placed on results from often fragile epidemiological studies, with major sources of bias ignored or totally underestimated. In contrast, far too little faith is placed on evidence that nonsmokers have very much lower exposure to tobacco smoke constituents than do smokers, and that smokers are much more exposed to ETS than nonsmokers.

The evidence that exposure to ETS increases the risk of developing heart disease is extremely unconvincing. Of the studies cited by Wells, some are based on unacceptably small numbers of cases, e.g., Garland et al. (1985) where only two deaths occurred in women married to never-smoking husbands, while the only two studies with substantial numbers of deaths are both open to question.

When referencing the Japanese prospective study, Wells uses Hirayama's 1984 report of a statistically significant positive trend in wife's age-adjusted risk according to husband's smoking, but does not comment on the fact that, in 1981, Hirayama reported no association whatsoever. As shown in Table 1, the

Table 1. Female relative risks for heart disease from passive smoking in Japanese study.

Follow-up period	Total cases	Husband's smoking habit		
		Non-smoker	Ex or <19/day	20+/day
1966-79	406	1	0.97	1.03
1980-82[1]	88	1	2.85	5.07
1966-82	494	1	1.10	1.30

[1] Estimated from 1966-79 data (Hirayama 1981) and from 1966-82 data (Hirayama 1984). The 1984 paper provided relative numbers of deaths as 118, 240, and 136.

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1966-79 and 1980-82 data are totally inconsistent and statistical tests confirm the highly significant ($p<0.001$) interaction between relative risk and period of follow-up. A possible explanation might be that the 1981 data, but not the 1984 data, were standardised additionally for occupation, but if this was important, why did Hirayama not standardise for occupation in 1984?

The Maryland prospective study (Helsing et al. 1988), which reported a 24% increase in heart disease risk in women, based on 988 deaths, and a 31% increase in men, based on 370 deaths, in relation to living with a smoker, has a number of features that should be considered when interpreting the data. No attempt was made to follow-up people moving outside Washington County, thus presumably missing large numbers of deaths. No dose-response relationship was reported. Adjustment for age, marital status, years of school and quality of housing had an enormous effect on relative risk, changing estimates from 1.17 to 1.31 in men and from 0.66 to 1.24 in women. No direct adjustment was made for household size, despite the fact that the larger the household, the more likely it is to contain a smoker. Furthermore, no direct adjustment was made for the possible correlation of household size with various coronary risk factors. Also, data were unavailable on a whole range of factors, such as diet and exercise, which might differ in families with and without smokers. In short, several potential confounders were apparently not controlled for.

Wells does not consider the problem of publication bias. This may be particularly acute for heart disease. After all, it is a vastly more common disease than lung cancer in nonsmokers, but the numbers of deaths in Wells' tables are only slightly greater. The possibility can surely not be excluded that other researchers, perhaps with much larger and better data bases, have looked at the relationship and found nothing.

The data for cancer other than the lung are even less convincing than for heart disease. In view of the much greater passive smoke exposure of smokers than nonsmokers, observations that nonsmokers exposed to passive smoking have increases in cancers at sites not increased in smokers seem to me to suggest that something is wrong with the epidemiological studies. And, indeed, the paper showing the strongest association (Sandler et al. 1985) is open to a number of serious criticisms (Lee 1985). Wells, however, remains content to include all epidemiological studies in his meta-analyses, regardless of quality, and attempts to explain obviously spurious relationships by an unsupported, and implausible hypothesis, involving an especially susceptible group of individuals who all die early if they smoke but die later by passive smoking if they do not. Mortality patterns for lung cancer in terms of age, dose, and duration of smoking are in fact well described by models involving no component for variation in susceptibility at all.

Wells' estimate of 3000 lung cancer deaths per year based on the epidemiological data contrasts with that of 12 by Arundel et al. (1987) based on extrapolation using relative amounts of particulate matter retained in the lung by nonsmokers and smokers. As I argue at length elsewhere (Lee 1987; 1988a; 1988b; 1989a; 1989b), it is far more plausible to conclude that the association observed between lung cancer and exposure to ETS arises predominantly because of bias than it arises because of a carcinogenic effect of such low doses of ETS.

Misclassification of smokers as nonsmokers is likely to be a major source of bias in most studies and is one for which Wells' correction is totally inadequate. He does not allow at all for the possibility of misclassified current typical regular smokers, whereas a recent summary of data from large studies shows an average rate of about 4% (Lee 1989a). Nor do his calculations take into account recent data (USSG 1989) showing much higher relative risks in active smokers than in older studies. Preliminary calculations based on these data suggest that the total number of lung cancers occurring in self-reported never smokers in the U.S. may have been substantially overestimated. Rather than 12 000 the figure may be nearer 8000. If reasonable corrections are made for misclassification, the figure of lung cancer deaths among actual never smokers may be less than 6000.

Wells considers his overall estimate of 46 000 deaths conservative. I disagree. When better data are available, it may prove to be about 46 000 too high.

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P. N. Lee Statistics and Computing Ltd.
Surrey, United Kingdom

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ISCHEMIC HEART DISEASE; RESPONSE TO LEE

Dear Editor:

Dr. P. Lee questioned the reasons for a discrepancy of my reports in 1981 and in 1984 on husbands' smoking and ischemic heart disease risk in nonsmoking wives.

The 1981 report was based on a 14 year follow-up (n=400) and the 1984 report was based on a 16 year follow-up (n=494) of nonsmoking wives. The relative risks of ischemic heart disease when husbands were nonsmokers, exsmokers, or daily smokers of 1-19 cigarettes and 20 or more cigarettes were 1.00, 1.06, and 1.18 (trend p : 0.061 not significant) in the 14 year follow-up; and 1.00, 1.10, and 1.31 (trend p : 0.019 significant) in the 1984 report.

Table 1. Ischemic heart disease mortality in women by age group, by occupation, and by husbands' smoking habit (patient herself a nonsmoker).

Husband's occupation	Husband's age group	Husband's smoking habit: Nonsmoker		Exsmoker 1-19/day		20+/day		Total	
Agricultural worker	40-49	8	2,502	25	5,941	17	3,636	50	12,079
	50-59	15	3,497	27	6,812	27	3,514	69	13,823
	60-69	36	4,084	79	6,845	27	2,152	142	13,081
	70-	5	323	11	446	2	89	18	858
	Total	64	10,406	142	20,044	73	9,391	279	39,841
Other	40-49	5	3,727	15	9,093	15	7,128	35	19,948
	50-59	11	4,294	29	8,830	23	6,306	63	19,430
	60-69	29	3,036	46	5,598	20	2,499	95	11,133
	70-	9	432	8	619	5	137	22	1,188
	Total	54	11,489	98	24,140	63	16,070	215	51,699
The weighted point estimate of rate ratio and test-based 90% confidence limits		1.00		1.11 (1.33 / 0.92)		1.36 (1.68 / 1.09)		Mantel extension chi	2.539
Mantel-Haenszel chi		-		0.882		2.331		One tail p value	0.00916
One-tail p value		-		0.18889		0.00988			

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